Effect of Acute Exposure to Loud Occupational Noise during Daytime on the Nocturnal Sleep Architecture, Heart Rate, and Cortisol Secretion in Healthy Volunteers

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Abstract: Effect of Acute Exposure to Loud Occupational Noise during Daytime on the Nocturnal Sleep Architecture, Heart Rate, and Cortisol Secretion in Healthy Volunteers: Batmanabane GITANJALI, et al. Department of Pharmacology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), India—Objectives—Noise is one of the commonest physical stressors to which industrial workers are exposed. Many workers complain of symptoms associated with a non-specific generalized stress response, including disturbed sleep. However, industrial workers may be exposed to more than one source of stress and it is not possible to completely attribute the disturbed nocturnal sleep and changes in heart rate to the effects of loud noise alone. This study was done to find out whether acute exposure of healthy individuals to loud occupational noise during the daytime would cause changes in their nocturnal sleep architecture, heart rate during sleep and serum cortisol levels. Methods—Baseline polysomnography was done on ten subjects who were exposed for eight hours either to continuous occupational background noise levels of >75dB(A), or a quiet environment. Sleep polysomnography was done on the night prior to and after exposure. Blood was collected for serum cortisol estimation at night prior to sleep and in the morning after waking up. Statistical analysis was done by repeated measures ANOVA with Tukey's post test. Results—The sleep efficiency was less than 80% and the total time spent in Rapid Eye Movement (REM) sleep, Slow Wave Sleep (SWS) and the REM onset latency were significantly decreased on the night after exposure to noise. There was a significant increase in stage shifts. The percentage fall in heart rate during sleep was decreased compared to the baseline values. The serum cortisol levels in the morning after exposure to noise was significantly increased. Conclusion—Workers exposed to loud background occupational noise react to the stress and show changes in nocturnal sleep architecture and heart rate which may be contributed to the exposure to noise. (J Occup Health 2003; 45: 146–152)

Key words: Occupational noise, Serum cortisol, Heart rate, Polysomnography, Sleep architecture, Sleep

Noise is one of the commonest occupational hazards of the modern world and there is evidence to support the increasing prevalence of high noise levels in the workplace. Loud noise has been shown to evoke physical, psychosocial and behavioural responses in animals and human beings which have been observed in the laboratory as well as in field studies. The non-auditory adverse effects of occupational noise exposure on cardiovascular functions, breathing, sleep, physical and mental health are now considered to be a serious cause for concern. Many industrial workers are exposed to loud noise throughout their workday and complain of disturbed sleep at night. Insomnia and disturbed sleep are two of the wide range of symptoms expressed by persons exposed to chronic noise.

Noise may act as a general, non-specific stressor and a recent study points to the fact that the neuroendocrine response of subjects exposed to low frequency ventilation noise are similar to other stressors. The sleep disturbance associated with stress has not been well-documented polysomnographically, predominantly due to its transient nature and the fact that not all persons who experience stress may respond with a detectable sleep disturbance. Persons working in an occupation with a loud background noise level may also be exposed to other types of stress. Hence field studies may not give a reliable indication of noise being a major stressor in order to attribute the disturbed nocturnal sleep to the effects of loud noise alone. A relatively wide range of individual differences in
Subjects and Methods

The study was a prospective experimental, crossover design conducted in the sleep disorders laboratory, Jawaharlal Institute of Postgraduate Medical Education and Research, Pondicherry, South India, a tertiary care hospital. All subjects were selected from Pondicherry by a purposive sampling method. Ten healthy male volunteers between 18–40 yr of age, working in shops as sales assistants were recruited. The background occupational noise level was 45 dB(A) and below. As it was to be a cross over interventional trial, only one group of subjects was needed. We excluded shift workers, those on medication for systemic and/or metabolic diseases, regular alcohol consumers, those with neuropathies and psychiatric illness, and those taking drugs to assist sleep. Clearance for the study was obtained from the Institute Research and Ethics Committees. Written informed consent was obtained from all volunteers.

The objective assessment of sleep was done by polysomnography and subjectively it was measured using a Sleep Evaluation Questionnaire10. The subjects slept in the Sleep Disorders Laboratory for a total of four consecutive nights. The first was an acclimatization night where the subjects slept in the laboratory with all electrodes fixed but no recording was done. The second night was for the baseline polysomnography recording which will be referred to as ‘baseline night’. On the morning following the baseline night, subjects were made to spend eight hours amidst the noise in a fully functioning weaving mill with sound levels >75 dB(A). They then had to report to the Sleep Disorders Laboratory for a polysomnography recording at night, henceforth referred to as ‘noise night’. On the following day, subjects were asked to spend eight hours in a room of approximate size, ventilation and temperature to the room in the mill. This was a quiet room, with sound pressure levels <45 dB(A). That night too, the subjects underwent a sleep polysomnography recording in the Sleep Disorders Laboratory which would be called ‘quiet night’. Subjects were made to spend a day in a quiet environment to rule out the stress which could occur due to being confined to one place. In order to prevent the carry-over effect of the daytime noise load, all subjects were randomized to spend Day 2 either in a quiet environment or in the mill. Random numbers were generated by a computer software programme. Five subjects spent day 2 in the mill and five spent day 3 in the mill and were then crossed over, hence, either night 3 or night 4 may be the noise night and vice versa.

Day 0

Night 1
(acclimatization - no recording)

Day 1 (preparation)

Night 2 (baseline sleep polysomnography)
‘baseline night’

Day 2 (in quiet place)

Night 3
(sleep polysomnography) ‘quiet night’

Day 3 (at rice mill)

Night 4
(sleep polysomnography) ‘noise night’

Day 4 (study ends)

The polysomnographic montage consisted of electroencephalography, electro-oculography, electromyography and electrocardiography (ECG)11. Standard leads were used for monitoring. Computerized polysomnography equipment (Alice 3), from Healthdyne Technologies (USA) was used for recording, and data was stored on magneto-optical disks with a capacity of 640MB for scoring later. Sleep scoring was done according to established criteria of Rechtschaffen and Kales11 using an epoch by epoch method. Length of each epoch was 30 s.

Heart rate was counted from the ECG tracing. Two consecutive epochs of each sleep stage from every sleep cycle during the course of the night was sampled and the averages taken. The average heart rate during sleep was the mean of the results obtained in all sleep stages. The awake levels were taken 10 min after the patient was hooked up for the study and lying in bed and before the lights were switched off.

In the morning, after the sleep study was completed, subjects were asked to rate their sleep quality during the previous night on three parameters, i.e. sleep onset latency, sleep continuity and sense of refreshed sleep. These were recorded on three separate 10 cm visual analogue scales (VAS) which recorded the poorest estimates of sleep at zero and the best estimates at 10 cm13. The VAS were translated into Tamil, a language with which all subjects...
were familiar. The subjects were allowed to go home after this. The physical activity of the subjects on Day 2 and Day 3 were almost identical.

Samples of blood to estimate serum cortisol were taken at 8.30 P.M on Nights 1, 2 and 3 and 7 A.M on Days 2, 3 and 4. The samples were centrifuged immediately and the serum collected and stored at −20 °C until analysis. Serum Cortisol was assayed by radioimmunoassay (RIA) using an RIA kit (Spectria®, Orion Diagnostica, Orion Corporation, Espoo, Finland). All samples were assayed together.

Sound levels at workplaces were measured with a Data Acquisition Sound Level Meter, Cygnet Inc., U.S.A., a hand held sound measuring device. The measurements at the mill and the room were made with the help of the field staff of the Department of Science Technology and Environment, Govt. of Pondicherry.

Results were fed into Microsoft Office Excel Spreadsheet. They were analysed using GraphPad Prism version 3.00 for Windows, GraphPad Software, San Diego, California USA. Repeated measures Analysis of Variance was done with Tukey's post test. A significance level of p<0.05 was chosen.

Results

The mean age (SD) of the subjects was 24.7 (2.28) yr, weight was 56.1 (4.45) kg, and height was 168 (7.5) cm. They all were employed for an average time of 6.6 (3.2) yr.

All polysomnography recordings in each subject were done on successive nights, except for one subject in whom it was done after a short gap of a couple of days (after the quiet night). This was because the subject lost his uncle and could not come to the sleep laboratory during the night. A total of ten subjects were studied, out of which all polysomnography records were recorded except one which was stored on a faulty disc and could not be retrieved. Therefore, all data included (for the polysomnography study and not for the cortisol estimation) are the results for nine subjects only.

The noise levels as recorded by a sound level meter were 87 dB(A) in the mill and 40.5 dB(A) in the room. There was no significant difference in the subjective measures of sleep quality between the various nights with all subjects expressing more than 80% satisfaction with the sleep quality on a VAS scale on all nights. The mean total REM time ± SD in the noise night was 58.0 ± 9.7 min which was significantly less (p<0.0001) compared to the 105.4 ± 14.8 min in the baseline night. The total time spent in SWS was also significantly less in the noise night 71.1 ± 7.3 compared to the baseline night 87.1 ± 13.7, though at a lower level of significance (p<0.05). The REM onset latency was 122.8 ± 24.0 min in the noise night and 88.0 ± 13.9 and 77.2 ± 12.4 min in the quiet and baseline nights respectively which was significantly different between noise and baseline nights (p<0.001) and also between quiet and baseline nights (p<0.01) respectively. No significant changes were seen for sleep onset latency and for Stage 1 and 2 of NREM sleep. The number of stage shifts (mean ± SD) in the noise night was 149.1 ± 23.2 and 77.3 ± 50.7 in the baseline night which was significantly higher (p<0.001). This increase

![Fig. 1. Hypnograms of the same subject of the baseline night (top panel), quiet night (intermediate panel) and noise night (bottom panel).](image-url)
Table 1. Mean heart rate (beats/min) during awake and sleep states in the volunteers

<table>
<thead>
<tr>
<th></th>
<th>Baseline night</th>
<th>Quiet night</th>
<th>Noise night</th>
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<tbody>
<tr>
<td>Awake</td>
<td>70.7 ± 2.37</td>
<td>61.9 ± 4.1*</td>
<td>70.3 ± 2.8</td>
</tr>
<tr>
<td>Sleep</td>
<td>62.0 ± 6.0#</td>
<td>69.5 ± 2.1</td>
<td>65.4 ± 2.8</td>
</tr>
</tbody>
</table>

All values are mean ± SD. n=9; ANOVA p<0.0001; Tukey’s post test. *: p<0.001; #: p<0.01
Baseline night is the control night. Quiet night refers to the night following the time spent in quiet surroundings during the daytime; Noise night refers to the night following the time spent in noisy surroundings during daytime. Values are mean ± SD (mm); n=9 subjects.

Table 2. Mean serum cortisol (nmol/l) in the volunteers before sleep and after waking up during the various interventions

<table>
<thead>
<tr>
<th></th>
<th>Morning samples</th>
<th>Night samples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>714.5 ± 266.3</td>
<td>226.2 ± 139.5</td>
</tr>
<tr>
<td>Quiet</td>
<td>775.1 ± 321.2</td>
<td>283.5 ± 179.2</td>
</tr>
<tr>
<td>Noise</td>
<td>985.0 ± 479.8*</td>
<td>323.6 ± 154.8</td>
</tr>
</tbody>
</table>

Values are mean ± SD; n=10; The samples were collected between 2,000 and 2,100 h at night and between 06.00 and 07.00 h in the morning after the subjects woke up. P<0.0001 by repeated measures ANOVA followed by Tukey’s post test. *: p<0.05 when compared to baseline.

Fig. 2. Percentage change in heart rate of each subject between wakefulness and sleep during the three nights. n=9 in each night. p<0.01; One way ANOVA. F=7.494; R squared=0.384.

Fig. 3. Serum cortisol values of ten subjects
BLnight: Baseline Night, BLmorn: Baseline Morning, Qnight: Quiet Night, Qmorn: Quiet Morning, Nnight: Noise Night,Nmorn: Noise Morning. Each point denotes the serum cortisol value for a single subject.

There were no significant differences in heart rate within the sleep stages between nights. However, significant differences between heart rates during wakefulness and during sleep were seen (One way ANOVA; p<0.0001) between stages, across nights. On subjecting the data to Tukey’s test significant differences were seen between the sleep and awake states in the baseline night (p<0.001), and in the quiet night (p<0.01). There were no significant differences (Table 1) between the awake and sleep states in the noise night, though values during sleep were lower. The percentage change in heart rate between wakefulness and sleep showed that there was very little change in the noise night (Fig. 2).

Mean serum cortisol levels showed significant diurnal variation on all days. However, statistically significant differences were seen between baseline night and noise night but not between baseline night and quiet night (Table 2). Figure 3 gives the individual serum cortisol values of each subject.
Discussion

The study proves that nocturnal sleep architecture is disturbed in healthy subjects who are exposed to loud occupational noise during the daytime and that this may be a direct consequence of stress. Surprisingly, the subjects did not feel any change in the quality of sleep which is a frequent complaint of workers in field studies. Therefore, there seems to be a mismatch between the objective and subjective findings with regard to sleep quality. Many studies have referred to this phenomenon before\(^\text{[12, 13]}\). There maybe critical levels of changes in the polysonomography parameters to be reached before such a polysonomography finding is translated as a subjective feeling of unrefreshed sleep\(^\text{[14]}\) or it may be due to the comfortable surroundings in the sleep laboratory which may negate any slight subjective feeling of poor sleep quality.

The decrease in REM sleep may explain some of the symptoms listed by workers exposed to noise stress, such as, changes in mood, difficulty in concentration, irritability, etc.,\(^\text{[15–17]}\) because REM sleep is said to be involved in modulation of mood\(^\text{[18]}\) and the maintenance of an organism’s capacity to sustain attention during wakefulness\(^\text{[19]}\). Though there is a decrease in SWS it may not be clinically significant considering the small difference between nights. A previous study on twelve healthy subjects exposed to loud industrial noise [85dB(A)] for twelve hours also showed some changes in sleep stages such as a decrease in REM time, shortened sleep cycles and an increase in deep sleep during the second sleep cycle\(^\text{[20]}\). The same researchers also reported that the pituitary responses to such noise stress were very much individualized and could not identify a trend in their responses\(^\text{[21]}\). This differs from our study in which there seems to be a consistency in the cortisol response among subjects. Though the subjects in our study too were exposed to loud industrial noise, the duration of exposure was equal to a normal working day of eight hours. The differences in sleep architecture of the previous study may be explained by the more prolonged stress and also the fact that the noise exposure continued almost until the bedtime of the subjects\(^\text{[20, 21]}\).

The increase in serum cortisol is seen only in the morning after the noise night and not in the evening after exposure to noise. This suggests a delayed cortisol response to stress. A previous study on workers exposed to loud noise also showed no increase in serum cortisol immediately following the noise exposure\(^\text{[22]}\). Noise stress has repeatedly been shown to increase cortisol levels. Though cortisol levels are high after quiet and noise interventions statistically significant differences are seen between baseline and noise nights only. Since all the subjects were exposed to identical conditions it could be presumed that loud noise per se contributed significantly to increased levels of cortisol. The reason why no differences were seen between the quiet and noise nights were perhaps due to a small degree of stress that could have been produced due to the subjects being constrained in a small space in the room during the quiet intervention.

Most blue collar workers in the industry have no control over their jobs. With rising unemployment, workers in India have no control over the type of work environment they can choose for themselves. This creates a sense of helplessness. In a previous study healthy human volunteers exposed to the stress of loud (100 dB) noise reported higher self-ratings of helplessness, lack of control, tension, stress, unhappiness, anxiety, and depression\(^\text{[23]}\). Lack of control over even a mildly aversive stimulus can produce alterations in mood as well as neuroendocrine and autonomic nervous system changes in healthy subjects\(^\text{[24]}\).

The heart rate normally falls during sleep by about 7–10% from the awake levels in all subjects and this fall is a result of the diminishing sympathetic activity and increasing parasympathetic tone\(^\text{[25, 26]}\). Workers exposed to loud noise levels have exhibited increases in heart rate and a sympathetic dominance\(^\text{[27, 28]}\). These measurements, however, were taken at the time of exposure to loud noise, whereas in our study they were done at night in a quiet atmosphere. To the best of our knowledge, there are no studies recording physiological variables at night, about four to five hours after the exposure to noise had ceased. As is evident from the individual changes in the fall in heart rate during sleep and the decrease in sleep efficiency below 80%, not all persons respond to loud noise in a similar fashion. The differential reactivity of rats to various types of stress has been demonstrated in an experiment wherein rats, subjected to noise stress, responded differently from those subjected to restraint stress\(^\text{[29]}\). It may, therefore, be considered that humans too may be responding to noise stress in a similar fashion, with some individuals responding with a disturbed sleep profile more severely than others. Mildly aversive stimuli such as noise have produced changes in cardiovascular reactivity and activation of the sympathetic nervous system\(^\text{[30]}\). The decrease in fall of heart rate suggests that this may be one of the factors involved. Previous work has shown that prolonged exposure to loud noise causes an increased stimulation of the sympatho-adrenal system and might lead to an abnormal response of the cardiovascular system\(^\text{[31, 32]}\). It may be speculated that, in a similar fashion, workers who are experiencing noise stress may respond by their heart rates not falling at night and may be more prone to develop cardiovascular morbidity. Psychological stress at the workplace have been shown to result in poor sleep quality measured subjectively\(^\text{[30]}\). In any given work environment, different people react differently some perceiving the working
conditions as highly stressful and others as not stressful\(^6\). There is some evidence pointing to the fact that loud occupational noise leads to an increase in cardiovascular morbidity\(^7\) but the exact factors contributing to this effect have not yet been worked out.

The decrease in the fall in the heart rate during sleep is also possibly linked to the neuroendocrine responses to stress since previous studies in animals\(^8\) and humans\(^9,10\) have shown that cardiovascular reactivity is linked to neuroendocrine reactivity possibly within the central nervous system.

This study proves that nocturnal sleep of subjects exposed to loud occupational noise during the daytime is adversely affected. It also shows the neuroendocrine link between loud occupational noise and stress and proves that the subjects react to this stress with an abnormal cardiovascular response as indicated by the decrease in the fall in heart rate during sleep. While these results need to be corroborated in the field, it may not be premature to say that the non-auditory adverse effects of loud occupational noise may have more insidious and far reaching consequences for the worker.

References

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